Acute type B aortic dissection in the absence of aortic dilatation

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Background: **Increasing aortic diameter is thought to be an important risk factor for acute type B aortic dissection (ABAD). However, some patients develop ABAD in the absence of aortic dilatation. In this report, we sought to characterize ABAD patients who presented with a descending thoracic aortic diameter <3.5 cm.**

Methods: **We categorized 613 ABAD patients enrolled in the International Registry of Acute Aortic Dissection from 1996 to 2009 according to the aortic diameter <3.5 cm (group 1) and ≥3.5 cm (group 2). Demographics, clinical presentation, management, and outcomes of the two groups were compared.**

Results: **Overall, 21.2% (n = 130) had an aortic diameter <3.5 cm. Patients in group 1 were younger (60.5 vs 64.0 years; *P* = .015) and more frequently female (50.8% vs 28.6%; *P* < .001). They presented more often with diabetes (10.9% vs 5.9%; *P* = .050), history of catheterization (17.0% vs 6.7%; *P* = .001), and coronary artery bypass grafting (9.7% vs 3.4%; *P* = .004). Marfan syndrome was equally distributed in the two groups. The overall in-hospital mortality did not differ between groups 1 and 2 (7.6% vs 10.1%; *P* = .39).**

Conclusions: **About one-fifth of patients with ABAD do not present with any aortic dilatation. These patients are more frequently females and younger, when compared with patients with aortic dilatation. This report is an initial investigation to clinically characterize this cohort, and further research is needed to identify risk factors for aortic dissection in the absence of aortic dilatation. (J Vasc Surg 2012;56:311-6.)**

Acute aortic dissection is a cardiovascular emergency which is associated with high mortality and morbidity.[1-4] Increasing aortic diameter is thought to be an important risk factor of acute aortic dissection and rupture,[5-8] and prophylactic descending thoracic aortic repair is generally recommended for an aortic diameter larger than 5.5 or 6.0 cm.[4-9] However, a recent International Registry of Acute Aortic Dissection (IRAD) study revealed that the majority of patients with acute type B aortic dissection (ABAD) appear to develop dissection in aortas smaller than this threshold.[10] Closer evaluation of this group suggested that a considerable number of patients present ABAD in the absence of aortic dilatation (Fig 1). It is currently unclear how many patients develop ABAD without aortic enlargement, who these patients are, and why they develop aortic dissection. Because these patients do not have any indications for prophylactic descending thoracic aortic repair prior to dissection, it is important to characterize this group. The purpose of this study was to investigate ABAD patients with a descending thoracic aortic diameter smaller than 3.5 cm.

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METHODS

Patient selection and data collection. IRAD is an ongoing multinational multicenter registry that enrolls patients with acute aortic dissection at 24 large referral centers; the inception and structure of IRAD has been described previously.[11] All patients presenting with ABAD that were enrolled in the IRAD registry between 1996 and September 2009 were selected for analysis. Only patients of whom the descending aortic diameter at presentation was available were included for this evaluation. In total, we identified 613 patients with ABAD who were included for analysis. Patients were categorized according to an aortic diameter <3.5 cm (group 1) and ≥3.5 cm (group 2), and the two groups were compared regarding demographics, clinical presentation, management, and outcomes.

The maximum descending aortic diameters were measured by computed tomography, transesophageal echocardiography, and/or magnetic resonance imaging at the time of presentation. If patients underwent multiple imaging

Fig 1. Acute type B aortic dissection (ABAD) in the absence of aortic dilatation.

studies, the study that reflected the largest aortic diameter was selected for analysis. The maximum aortic diameter was measured at cross-sectional images perpendicular to the long axis of the descending aorta. In IRAD, all aortic diameter measurements were obtained after aortic dissection had occurred.

Statistical analysis. Data analysis was performed with the use of SPSS statistical analysis software (SPSS Inc, Chicago, Ill). The χ^2 test was used for comparing categorical variables between patients with an aortic diameter <3.5 cm and ≥ 3.5 cm; the Student *t*-test was used for comparing continuous variables between both diameter groups. Summary statistics are presented as frequencies and percentages for categorical variables and mean ± standard deviation for continuous variables. In all cases, missing data were not defaulted to negative, and denominators reflect only cases reported. A *P* value $<.05$ was considered significant. The authors had full access to and took full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

RESULTS

Aortic measurements. The mean aortic diameter at the level of ABAD was 4.40 ± 1.35 cm (median, 4.1 cm; range, 2.1-13.0 cm). ABAD in the absence of aortic dilatation, defined as a descending aortic diameter smaller than 3.5 cm, was present in 21.2% (n = 130) of patients in IRAD. The mean aortic diameter in group 1 was 2.97 ± 0.32 cm, compared with 4.79 ± 1.26 cm in group 2 ($P <$ .001). In females, the mean aortic diameter in the overall group was 4.30 cm, compared with 4.46 cm in males ($P =$ .199).

Demographics and patient history. The mean age of ABAD patients in IRAD was 63.9 ± 14.0 years, and 67.7% were males. The mean age of patients without aortic dilatation was 60.5 years, compared with 64.0 years in patients with an aortic diameter larger than 3.5 cm ($P =$ .015). Among patients in group 1, 50.8% were females, compared with 28.6% among patients in group 2 ($P <$ .001). After stratification for aortic diameter, ABAD patients were predominantly males in all aortic diameter categories, except for those in the aortic diameter categories below 3.5 cm (Fig 2), in which the distribution of males and females was approximately equal. Biometric characteristics such as height and weight did not differ significantly between patients with and without aortic enlargement (Table I). Patients without aortic dilatation were more likely to report a history of diabetes mellitus (10.9% vs 5.9%; $P =$ .050), while pre-existing hypertension (73.6% vs 81.5%; $P =$ .050) was less common in this cohort. Marfan syndrome and bicuspid aortic valve were present in 4.3% and 1.9% of all ABAD patients, respectively, and prevalences did not differ between both diameter groups. Prior coronary artery bypass grafting (CABG) and cardiac catheterization were more frequently represented in patients without aortic dilatation. In this group, an iatrogenic cause of dissection was also more present (5.6% vs 1.1%; $P =$ .002; Table I).

Presentation and diagnostic findings. ABAD patients without aortic dilatation presented less frequently with chest pain (64.0% vs 73.4%; $P =$ .038) or abdominal pain (31.1% vs 42.1%; $P =$ .028) than patients with a larger aortic diameter. Syncope at presentation was more common in patients without aortic enlargement (7.2% vs 2.3%; $P =$ .007). Complicated ABAD, defined as presence of

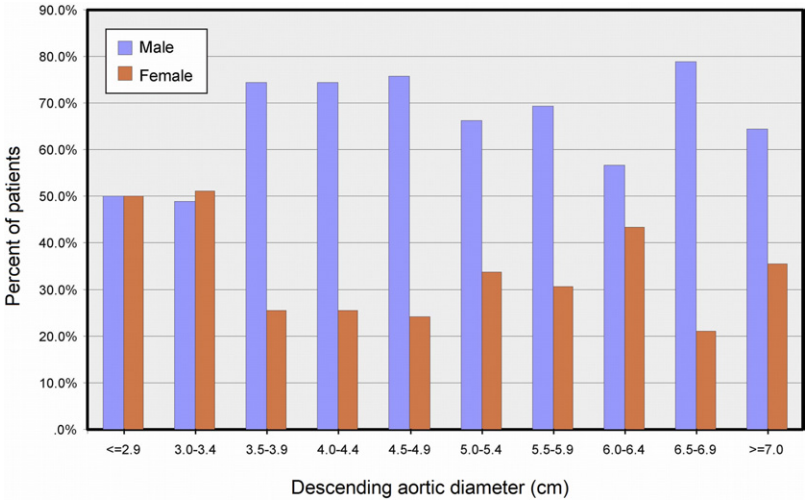

Fig 2. Distribution of gender in different aortic diameter groups. Acute type B aortic dissection (ABAD) patients in International Registry of Acute Aortic Dissection (IRAD) were predominantly males in all aortic diameter categories, except for those in the aortic diameter categories below 3.5 cm, in which the distribution of males and females was approximately equal.

Table I. Demographics and clinical history

	Aortic diameter <3.5 cm		*Aortic diameter ≥3.5 cm*		
	n	*%*	*n*	*%*	P *value*
	130	21.2	483	78.8	—
Demographics					
Age (years)	60.5	±14.8	64.0	±13.3	.015
Female	66	50.8	138	28.6	<.001
Biometrics					
Height (m)	1.68	±14.6	1.69	±12.1	.758
Weight (kg)	79.1	±21.5	87.1	±20.7	.188
Body mass index (m/kg^2)	28.1	±6.7	29.5	±5.7	.486
History					
Marfan syndrome	6	4.6	20	4.2	.840
Bicuspid aortic valve	2	1.9	7	1.9	.966
Hypertension	95	73.6	391	81.5	.050
Atherosclerosis	42	32.6	165	34.8	.633
Diabetes	14	10.9	28	5.9	.050
Prior aortic repair	11	8.9	73	15.5	.058
Prior coronary artery bypass grafting	12	9.7	16	3.4	.004
Prior catheterization	18	17.0	24	6.7	.001
Iatrogenic dissection	7	5.6	5	1.1	.002

Data regarding height were available for 8.2% (n = 50), weight for 11.1% (n = 68), body mass index for 8.0% (n = 49), and body surface area for 8.0% of patients (n = 49).

malperfusion syndromes, aortic rupture, recurrent or refractory pain, and/or refractory hypertension, was present in 31.5% of patients without aortic dilatation, compared with 40.6% of patients with an aortic diameter ≥3.5 cm ($P = .060$; Table II). A completely normal chest X-ray was seen in 43.5% in group 1 compared with 20.8% in group 2 ($P < .001$; Table III). Additional imaging showed a patent false lumen in 56.7% of patients without aortic dilatation and in 45.5% of the patients with an aortic diameter >3.5 cm ($P = .042$).

Management and outcomes. Medical management alone was offered to 69.7% of all patients. Endovascular methods, either endovascular stenting or fenestration, were more frequently offered to ABAD patients without aortic dilatation (13.8% vs 4.2%; $P < .001$), while surgical management was more common among patients with an aortic diameter >3.5 cm (Table IV). The overall in-hospital mortality was 9.6% (n = 53), and the mortality did not differ significantly between both diameter groups (7.6% vs 10.1%; $P = .398$). The mortality rate after endovascular management of ABAD patients with a normal aortic diameter was 23.5% (4 of 18), while no patients with an aortic diameter ≥3.5 cm died after endovascular management ($P = .040$).

Table II. Symptoms and signs of aortic dissection

	Aortic diameter <3.5 cm		*Aortic diameter ≥3.5 cm*		
	n	*%*	*n*	*%*	P *value*
Presentation					
Chest pain	80	64.0	348	73.4	.038
Abdominal pain	38	31.1	196	42.1	.028
Shock	2	1.6	5	1.1	.628
Syncope	9	7.2	11	2.3	.007
Complications	41	31.5	196	40.6	.060
Limb ischemia	9	7.6	36	8.1	.869
Any pulse deficit	22	18.8	66	15.5	.389
Visceral ischemia	8	6.7	16	3.6	.132
Acute renal failure	14	11.9	52	11.6	.938
Spinal cord ischemia	1	0.9	11	2.5	.290
Periaortic hematoma	11	9.4	79	18.2	.023
Recurrent pain	11	44.0	51	33.8	.322
Refractory pain	1	4.0	20	13.2	.187
Refractory hypertension	1	4.0	10	6.6	.616

DISCUSSION

Increasing aortic diameter is thought to be an important risk factor for acute aortic dissection and rupture[5-8]; however, about one-fifth of ABADs occur in patients without aortic enlargement. In this analysis, aortic diameter measurements were obtained after aortic dissection had occurred, and therefore the original diameter may have been even smaller. We observed remarkable differences between the two patient groups; most notably that women accounted for half of the ABAD patients with a normal diameter, while the majority of the type B dissections in enlarged aortas consisted of male patients. Male gender is a well-known risk factor for the development of various cardiovascular diseases, including acute aortic dissection, and approximately 65% to 75% of admitted patients with ABAD are males.[12-16]

It remains a mystery why patients sustaining ABAD in the absence of aortic dilatation are more frequently females. Women are typically smaller in size than men, and the average female aorta may be slightly smaller than the average male aorta. Consequently, a logical hypothesis for the increased proportion of females among ABAD patients with a normal aortic diameter could be that the female aorta may dissect at a smaller diameter, because a 3-cm aortic diameter may be considerably increased compared with the normal aortic measurements in females. However, in the overall IRAD cohort, the mean aortic diameter of ABAD patients did not differ substantially between males and females (4.5 cm vs 4.3 cm). In addition, patients' height did not differ between those presenting with and without aortic dilatation.

The increased proportion of females in the group without aortic enlargement may have other explanations beyond anatomical reasons. The natural history of thoracic aortic disease has not been well defined yet,[17,18] and in particular the influence of gender remains currently controversial. In contrast to abdominal aortic aneurysms, for which men are affected predominantly, the prevalence of thoracic aortic aneurysm (TAA) is thought to be more equal in women and men.[19] Women, however, typically present with aortic aneurysms and dissections at a greater age than men.[12,19,20] Moreover, female gender appears to be a risk factor for aortic aneurysm rupture.[19-21]

Another remarkable finding was that ABAD patients without aortic dilatation were on average 3.5 years younger, which is especially interesting since women generally present with ABAD at a higher age.[12] It is unclear why ABAD without aortic dilatation occurred at a younger age, but this may suggest a genetic or familial predisposition in some of these patients. Coady and colleagues have investigated familial TAA, which accounted for at least 20% of patients with TAA, after excluding Marfan patients.[22,23] They compared these nonsyndromic familial TAA patients with sporadic TAA patients and Marfan syndrome, and found that patients with familial TAA were about 7 years younger than patients with sporadic TAA. Interestingly, the aortic growth rate was highest for the familial TAA group in their analysis.[22,23] It is possible that a similar unknown familial or genetic predisposition exists in some young ABAD patients without aortic dilatation.

Marfan syndrome is an important risk factor for aortic dissection, especially in young patients,[5,7] and thresholds for prophylactic aortic replacement are typically lower for this specific patient group.[5-7,24-26] In ABAD patients without aortic dilatation, we did not find an increased prevalence of Marfan, perhaps because Marfan syndrome patients typically suffer from aortic root enlargement and dissection, instead of descending thoracic aortic disease.[24-26] Other inherited disorders, including Ehlers-Danlos syndrome type IV, Turner syndrome, and other less well-known connective tissue disorders, are known to affect the integrity of the arterial wall, which could result in aortic dissection.[4,27-31] IRAD does not contain data regarding inherited disorders other than Marfan syndrome, the most common connective tissue disease, and it is possible that the normal-diameter group had an increased prevalence of known, or currently unknown, connective tissue disorders. The genes that predispose to such connective tissue disorders and familial forms of thoracic aortic disease have only recently begun to be identified. Research initiatives, such as the National Registry of Genetically Triggered Thoracic Aortic Aneurysms and Cardiovascular Conditions,[32] have been established to further investigate genetically triggered thoracic aortic disease, and these registries may provide more insights about patients who develop ABAD in the absence of aortic dilatation.

Furthermore, iatrogenic dissection was more common among patients that suffered ABAD without aortic dilatation. It remains unclear why iatrogenic dissection was more common among this group, although a history of CABG and catheterization was more frequently present among ABAD patients without aortic dilatation. If iatrogenic aortic dissection occurs, it often arises during cardiac surgery or catheterization, and the incidence of iatrogenic type A

Table III. Diagnostic findings of aortic dissection

	Aortic diameter <3.5 cm		*Aortic diameter ≥3.5 cm*		
	n	*%*	*n*	*%*	P *value*
Chest X-ray findings					
Normal	50	43.5	90	20.8	<.001
Widened mediastinum	39	34.2	210	49.4	.004
Abnormal aortic contour	33	29.5	211	50.7	<.001
Additional imaging findings					
Intramural hematoma	27	20.9	89	18.6	.547
Patent false lumen	59	56.7	171	45.5	.042
Partial false lumen thrombosis	31	29.8	144	38.3	.111
False lumen thrombosis	14	13.5	61	16.2	.492
Mean descending aorta diameter	2.97	±0.32	4.79	±1.26	<.001
Entry tear located at left subclavian artery	76	59.4	259	55.1	.388
Entry tear located at descending aorta	22	17.2	113	24.0	.100

Table IV. In-hospital management and mortality

	Aortic diameter <3.5 cm		*Aortic diameter ≥3.5 cm*		
	n	*(%)*	*n*	*(%)*	P *value*
Definitive management					
Surgery	21	16.2	126	26.2	.017
Medical	91	70.0	335	69.6	.938
Endovascular	18	13.8	20	4.2	<.001
In-hospital mortality	9	7.6	44	10.1	.398
Surgery	1	5.0	19	16.1	.306
Medical	4	4.9	25	8.4	.286
Endovascular	4	23.5	0	0.0	.040

dissection during cardiac surgery or catherization is approximately 0.20% and 0.024%, respectively.[33,34]

Although investigating the management and outcomes was not the purpose of this study, we observed that endovascular stenting and fenestration were more frequently offered to patients with a normal aortic diameter, while those with aortic dilatation more frequently underwent surgery, perhaps because large dissected aortas more frequently have inadequate aortic landing zones for stenting. The in-hospital mortality after endovascular management appeared to be higher for the patients with normal aortic diameters than for those with aortic dilatation; however, the number of patients treated with endovascular management was too small to draw strong conclusions.

The IRAD database contains the largest series of patients with ABAD, which provided a unique opportunity to clinically characterize this subset of patients without aortic dilatation. As with all observational studies, this investigation has limitations that must be kept in mind when the data are interpreted. The aortic size measurements were obtained after acute dissection, and thus the actual aortic diameters prior to the event may be even smaller. IRAD does not have a disease-free control group, and therefore the data cannot be used to investigate which individuals with a normal aortic diameter are at risk for developing ABAD.

CONCLUSIONS

About one-fifth of patients with ABAD do not have any aortic dilatation prior to aortic dissection. This report is an initial attempt to clinically characterize this cohort, which revealed that these patients are younger and more frequently women. Further research is needed to explore the molecular and genetic predisposition of these patients.

AUTHOR CONTRIBUTIONS

Conception and design: ST, FJ
Analysis and interpretation: ST, FJ, KE
Data collection: ST, FJ, JF, GU, VR, HP
Writing the article: ST, FJ, KE
Critical revision of the article: JF, GU, FM, BM, VR, HP
Final approval of the article: ST, FJ, JF, GU, FM BM, VR, HP, KE
Statistical analysis: FJ
Obtained funding: KE
Overall responsibility: ST

REFERENCES

1. Anagnostopoulos CE, Prabhakar MJ, Kittle CF. Aortic dissections and dissecting aneurysms. Am J Cardiol 1972;30:263-73.
2. Wheat MW Jr. Acute dissecting aneurysms of the aorta: diagnosis and treatment. Am Heart J 1979;1980:373-87.
3. Olsson C, Thelin S, Ståhle E, Ekbom A, Granath F. Thoracic aortic aneurysm and dissection: increasing prevalence and improved outcomes reported in a nationwide population-based study of more than 14,000 cases from 1987 to 2002. Circulation 2006;114:2611-8.
4. Golledge J, Eagle KA. Acute aortic dissection. Lancet 2008;372:55-66.
5. Elefteriades JA. Natural history of thoracic aortic aneurysms: indications for surgery, and surgical versus nonsurgical risks. Ann Thorac Surg 2002;74:S1877-80.
6. Coady MA, Rizzo JA, Hammond GL, Kopf GS, Elefteriades JA. Surgical intervention criteria for thoracic aortic aneurysms: a study of growth rates and complications. Ann Thorac Surg 1999;67:1922-6.
7. Davies RR, Goldstein LJ, Coady MA, Tittle SL, Rizzo JA, Kopf GS, et al. Yearly rupture or dissection rates for thoracic aortic aneurysms: simple prediction based on size. Ann Thorac Surg 2002;73:17-27.

8. Lobato AC, Puech-Leão P. Predictive factors for rupture of thoracoabdominal aortic aneurysm. J Vasc Surg 1998;27:446-53.
9. Svensson LG, Kouchoukos NT, Miller DC, Bavaria JE, Coselli JS, Curi MA, et al. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. Ann Thorac Surg 2008;85:S1-41.
10. Trimarchi S, Jonker FH, Hutchison S, Isselbacher EM, Pape LA, Patel HJ, et al. Descending aortic diameter of 5.5 cm or greater is not an accurate predictor of acute type B aortic dissection. J Thorac Cardiovasc Surg 2011;142:e101-7.
11. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. The international registry of acute aortic dissection (IRAD): new insights into an old disease. JAMA 2000;283:897-903.
12. Nienaber CA, Fattori R, Mehta RH, Richartz BM, Evangelista A, Petzsch M, et al. Gender-related differences in acute aortic dissection. Circulation 2004;109:3014-21.
13. Neya K, Omoto R, Kyo S, Kimura S, Yokote Y, Takamoto S, et al. Outcome of Stanford type B acute aortic dissection. Circulation 1992; 86:II1-7.
14. Elefteriades JA, Hartleroad J, Gusberg RJ, Salazar AM, Black HR, Kopf GS, et al. Long-term experience with descending aortic dissection: the complication-specific approach. Ann Thorac Surg 1992;53:11-20.
15. Umaña JP, Lai DT, Mitchell RS, Moore KA, Rodriguez F, Robbins RC, et al. Is medical therapy still the optimal treatment strategy for patients with acute type B aortic dissections? J Thorac Cardiovasc Surg 2002; 124:896-910.
16. Estrera AL, Miller CC, Goodrick J, Porat EE, Achouh PE, Dhareshwar J, et al. Update on outcomes of acute type B aortic dissection. Ann Thorac Surg 2007;83:S842-5.
17. Ince H, Nienaber CA. Etiology, pathogenesis and management of thoracic aortic aneurysm. Nat Clin Pract Cardiovasc Med 2007;4: 418-27.
18. Isselbacher EM. Thoracic and abdominal aortic aneurysms. Circulation 2005;111:816-28.
19. Clouse WD, Hallett JW Jr, Schaff HV, Gayari MM, Ilstrup DM, Melton LJ III. Improved prognosis of thoracic aortic aneurysms: a population-based study. JAMA 1998;280:1926-9.
20. Mureebe L, Egorova N, McKinsey JF, Kent KC. Gender trends in the repair of ruptured abdominal aortic aneurysms and outcomes. J Vasc Surg 2010;51:9S-13S.
21. Brown LC, Powell JT. Risk factors for aneurysm rupture in patients kept under ultrasound surveillance. UK Small Aneurysm Trial participants. Ann Surg 1999;230:289-96.
22. Coady MA, Davies RR, Roberts M, Goldstein LJ, Rogalski MJ, Rizzo JA, et al. Familial patterns of thoracic aortic aneurysms. Arch Surg 1999;134:361-7.
23. Albornoz G, Coady MA, Roberts M, Davies RR, Tranquilli M, Rizzo JA, et al. Familial thoracic aortic aneurysms and dissections–incidence, modes of inheritance, and phenotypic patterns. Ann Thorac Surg 2006;82:1400-5.
24. Treasure T. Cardiovascular surgery for Marfan syndrome. Heart 2000; 84:674-8.
25. Dean JC. Management of Marfan syndrome. Heart 2002;88:97-103.
26. Gott VL, Greene PS, Alejo DE, Cameron DE, Naftel DC, Miller DC, et al. Replacement of the aortic root in patients with Marfan's syndrome. N Engl J Med 1999;340:1307-13.
27. Loeys BL, Schwarze U, Holm T, Callewaert BL, Thomas GH, Pannu H, et al. Aneurysm syndromes caused by mutations in the TGF-beta receptor. N Engl J Med 2006;355:788-98.
28. Pepin M, Schwarze U, Superti-Furga A, Byers PH. Clinical and genetic features of Ehlers-Danlos syndrome type IV, the vascular type. N Engl J Med 2000;342:673-80.
29. Elefteriades JA. Perspectives on diseases of the thoracic aorta. Adv Cardiol 2004;41:75-86.
30. Groenink M, Rozendaal L, Naeff MS, Hennekam RC, Hart AA, Van Der Wall EE, et al. Marfan syndrome in children and adolescents: predictive and prognostic value of aortic root growth for screening for aortic complications. Heart 1998;80:163-9.
31. Mukherjee D, Eagle KA. Aortic dissection–an update. Curr Probl Cardiol 2005;30:287-325.
32. Eagle KA, GenTAC Consortium. Rationale and design of the national registry of genetically triggered thoracic aortic aneurysms and cardiovascular conditions (GenTAC). Am Heart J 2009;157:319-26.
33. Januzzi JL, Sabatine MS, Eagle KA, Evangelista A, Bruckman D, Fattori R, et al. Iatrogenic aortic dissection. Am J Cardiol 2002;89:623-6.
34. Jonker FH, Schlosser FJ, Indes JE, Sumpio BE, Botta DM, Moll FL, et al. Management of type A aortic dissections: a meta-analysis of the literature. Ann Thorac Surg 2010;89:2061-6.

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